ARRHYTHMIAS AND ATRIOVENTRICULAR CONDUCTION DEFECTS AFTER MYOCARDIAL INFARCTION

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ARRHYTHMIAS AND ATRIOVENTRICULAR CONDUCTION DEFECTS AFTER MYOCARDIAL INFARCTION

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I

INTRODUCTION

Arrhythmias

Arrhythmias which arise secondary to myocardial infarction are an important cause of increased morbidity and mortality in the immediate post-infarction period. Such arrhythmias increase mortality from between 6.6 per cent to 21 per cent.[16,22] Indeed, some reports indicate that over 50 per cent of all deaths from myocardial infarction are caused by arrhythmias without any specific cause of death apparent at autopsy.[31] Ventricular fibrillation and standstill are most often implicated as the cause of sudden death. With the development and more widespread use of cardiac monitors and electronic devices for stimulating the heart, much interest has been directed recently towards earlier detection and treatment of post-infarction arrhythmias.

Cardiac Monitoring

The results of Spann et al.[28] with constant cardiac monitoring of 30 patients have revealed a higher incidence of arrhythmias than has been recorded in the past. It was their impression that "...70 per cent of the documented arrhythmias would have gone unnoticed without the aid of electronic monitoring." The incidence of arrhythmias in past studies has ranged from a low of 14.3 per cent[20] to a high of 73.2 per cent[15] where disturbances of rate were also included.

Electrical Stimulation

Until about 13 years ago the general treatment of cardiac arrhythmias relied basically on the use of the standard digitalis, quinidine,

procaine amide, sympathomimetic and parasympatholytic preparations. In 1952, Zoll published a review of his experiences with 2 patients who sustained ventricular standstill using a technique for artificial activation of the heart with metallic electrodes placed on the chest wall.[32] Subsequent improvement on methodsfor external cardiac stimulation and countershock,[34] and both short and long-term direct electric cardiac stimulation[33] have contributed significantly to the control and management of otherwise fatal cardiac arrhythmias. The most recent developments in the field of electronic stimulation of the heart have focused on the development of methods for permanent activation of the heart in complete heart block and Stokes-Adams attacks.[2,5,17] Lillehei et al.[19] first utilized direct myocardial pacing for complete heart block complicating surgery on septal defects. They noted that, "of the first seven patients with septal defects in whom this complication of open-heart surgery developed in 1954-55, all died in the immediate post-operative interval despite the use of epinephrine, ephedrine, atropine, sodium lactate and the external electric pacemaker." It was their opinion that mortality could be appreciably reduced by repetitive electrical stimuli of small magnitude delivered by an electrode implanted in the myocardium. At the time of their publication in 1960, patients had already been followed for up to 15 months with this equipment in place.

The Cathode Pacemaker

The development by Furman et al.[12] of a catheter electrode pacemaker has further expanded the spectrum of direct myocardial stimulation. The catheter pacemaker does not require thoracotomy for placement as it can be introduced into the right ventricle through one of the external

jugular veins. As of 1960, Furman et al. had followed 25 patients, the longest period of treatment being 16 months.[18] Landegren and Biorck treated 35 patients this way during 1962 with all but 2 patients alive as of January, 1963.

The ease with which the cathode pacemaker can be threaded into the right ventricle has excited interest in its use for acutely ill patients who could not tolerate an extensive surgical procedure as was required for direct myocardial stimulation in the past. Specifically this pacemaker has been used in patients with acute myocardial infarction complicated by asystole or heart block. Samet et al.[27] have reported on the use of the cathode pacemaker in 4 patients with acute myocardial infarction complicated by complete heart block. They observed dramatic clinical improvement in all 4 patients with the conversion of 2 to sinus rhythm. Two patients died, however, one of recurrent infarction and one from an intracerebral hemorrhage following anticoagulation. It was their opinion that "the cardiac catheter electrode pacemaker is probably the simplest and safest to employ for a relatively short term in the patient with acute myocardial infarction when external pacemaking is either intolerable or ineffective and when drug therapy is ineffective." DeSanctis et al.[10] indicated satisfactory results in their series of 16 patients paced for short periods of time. Thirteen of these 16 patients were still alive at the time of their respective publications. Bruce et al.[3] experienced only 1 death in 7 patients who were paced for 2 to 6 day periods. It was their feeling that the monitoring of patients with acute myocardial infarction and the immediate availability of the cathode pacemaker could significantly reduce mortality resulting from

A-V block or asystole. In addtion, the use of the cathode pacemaker need only be a temporary measure, as Penton et al.[24] have shown that it is exceedingly rare to see permanent heart block secondary to myocardial infarction in those patients who have survived and have previously had a normal sinus rhythm.

Heart Block Complicating Myocardial Infarction

The incidence of complete heart block complicating myocardial infarction is not high. Master et al.[21] reported an incidence of from 0.7 per cent to 10 per cent in 1500 cases of coronary artery occlusion with an average of 1.5 per cent. Although complete heart block is an uncommon complication of myocardial infarction, it does, however, carry with it a high mortality. Most studies quote a mortality of over 50 per cent with a range of between 43 per cent[21] and 100 per cent[22] before the use of electronic pacing. This is in sharp contrast to the experience of Bruce et al.[3] who found a mortality of only 14 per cent with use of the cathode pacemaker.

The incidence of incomplete or partial heart block complicating myocardial infarction has not been as widely studied as that of complete heart block. Indeed, Campbell[4] has found it to be very rare and a much less stable rhythm than complete heart block although it may persist in a few patients for a number of years. The incidence of lower grades of block has ranged from between 1 per cent to 12 per cent.[25] Both Master et al.[21] and Cohen et al.[6] felt that first degree block did not affect prognosis. Rosenbaum et al.,[26] however, did not separate their series into partial and complete heart block, for it was their experience that " patients showing one of these alterations commonly were found to

show the other at some time later." Indeed, of 7 patients determined to have some degree of A-V block, 2 went through all 3 degrees of block with eventual recovery; 2 showed complete block, 1 with reversion to a 2nd degree block and 1 to a 1st degree block; 1 with an initial 2nd degree reverted to a 1st degree mechanism. It should be emphasized, however, that mortality for those patients who develop only lower grades of A-V block is, in many series, lower than for those patients with complete block. In those series that do specifically distinguish between partial and complete block, comparable mortality figures have shown a death rate of 20 per cent for partial block as compared to 80 per cent for complete block,[15] and in another series 35 per cent as compared to 100 per cent.[22]

The Present Study

The present study has been designed to answer a number of questions about patients who develop A-V heart block complicating myocardial infarction as compared to patients who develop tachyarrhythmias and a "control" group who develop no arrhythmia. Four types of questions will be investigated:

1) What is the relative incidence of A-V block as compared to tachyarrhythmias and the "control" group?

2) What are the distinguishing features of the group with A-V block which may help to predict its occurrence? (This will be investigated with regard to the incidence prior to the present myocardial infarction of previous myocardial infarction, congestive heart failure, hypertension, antecedent arrhythmia, digitalization, and the location of the present infarct.)

3) What is the prognostic significance of A-V block in relation to the degree of block, secondary

complications, mortality, and the incidence of sudden death?

4) What are the chances for increased salvage of patients with A-V block with the use of cardiac monitoring and electrical stimulation?

II

METHODOLOGY

Selection of the Population

The population for study has been selected from the diagnostic index of the Grace-New Haven Community Hospital private and ward services. The available charts of all patients coded as having had a myocardial infarction from April 1, 1963 to March 31, 1964, were reviewed using the Standard Nomenclature of Diseases and Operations published by the American Medical Association. This included charts coded under the 3 diagnostic indices 430-516.7, 430-515.7, and 430-512.7 which signify respectively, 1) infarction of the myocardium due to arteriosclerotic coronary thrombosis, 2) infarction of the myocardium from coronary thrombosis due to arteritis, and 3) infarction of the myocardium due to embolism. The greatest percentage of patients were coded in the first group. Two hundred and twenty-six patients had myocardial infarction as the primary diagnosis; 105 as the second diagnosis; 37 as the third diagnosis, and 13 as the fourth diagnosis. Of the total of 381 patients coded as having had a myocardial infarction during the period outlined above, 300 consecutive cases were selected as the population for study. Only patients admitted with a presumptive diagnosis of myocardial infarction were included. This excluded patients who were found to have sustained myocardial infarction subsequent to admission to the hospital for other reasons whether surgical or diagnostic. The charts of a small group of the 381 patients were not available for review at this time.

Collection and Tabulation of Data

A form for the extraction of pertinent data from the hospital charts was designed and pre-tested on the first 20 consecutive cases. The form was developed to obtain 4 types of data pertaining to: 1) demographic distribution, 2) cardiac status prior to the present infarction, 3) factors relevant to the overall hospital course, and 4) factors relevant to the development of cardiac arrhythmias. Pertinent data was then organized for compilation and tabulation of demographic factors and study of the questions outlined above.

The possibility of statistical analysis of the data was investigated using the chi-square formulation and the formula for the difference between 2 binomial proportions. This analysis did not prove to be pertinent, however, because of the small numbers involved in individual categories and the inability to obtain precise statistical controls in the clinical situation under study.

III

DESCRIPTIVE DATA

Total Population

Of the total population of 300 patients, 202 were males and 98 females. There were only 8 Negroes included in the entire group. The median age for the entire population was 63 years with a range of from 30 to 92 years. The median age for males was 60.5, while the median age for females was 70 years. Forty-six per cent of the males were under 60 years of age while 22 per cent of the females were under 60 years of age. Only 22 per cent of the males were over 70 years of age while 43 per cent of the females were over 70 years of age. The mortality for the entire population was 31 per cent with an incidence of 94 deaths in 300 myocardial infarctions. The mortality for males was only 23 per cent while that for females was 38 per cent. The incidence of sudden death without shock, congestive heart failure, or embolization was 16 of 300 patients or approximately 5 per cent. The median length of stay in the hospital for those patients who did not die was 21 days with a range of 3 to 65 days.

Table I outlines the previous cardiac history for the entire population prior to the present admission in relation to previous myocardial infarction, congestive heart failure, hypertension, arrhythmias and digitalization.

TABLE I

CARDIAC HISTORY

Previous M.I.	NO.	% of Total
1	63	21
More than 1	12	4
Questionable	7	2
Cardiac Status		
CHF	71	24
Hypertension	108	36
Antecedent Arrhythmia	36	12
Medications		
Digitalis	73	24

Table II shows the relative incidence of the various locations of infarctions for 195 patients for whom there was electrocardiographic evidence of infarction. This represents 65 per cent of the entire population.

TABLE II

Location of Infarct	NO.	% of Total
Anterior-lateral	50	26
Anterior-septal	49	25
Posterior-diaphragmatic	86	44
Posterior-lateral	8	4
Mixed anterior and posterior	2	1

General morbidity in the entire population is outlined in Table III which depicts the incidence, secondary to myocardial infarction, of the complications of congestive heart failure, shock, embolization, and syncope.

TABLE III

Morbidity	NO.	% of Total
Congestive Heart Failure	103	34
Shock	87	29
Embolization	19	6
Syncope	8	3

The incidence of the use of those drugs which are known either to produce disturbances of rate and rhythm or are used to treat these disturbances is outlined in Table IV for the entire population.

TABLE IV

Drugs	NO.	% of Total
Digitalis	143	48
Procaine Amide	11	4
Quinidine	23	8
Diuretics	76	25
Atropine	12	4
Sympathomimetics	78	26
Steroids	10	3

Of the total population of 300 patients, 134 were found to have no evidence of rhythm or conduction abnormality during hospitalization. In addition, 45 patients were found to have evidence of premature ventricular or atrial beats without another associated arrhythmia. The remaining 121 patients had 1 or more abnormalities in rhythm and conduction. Excluding the group with evidence only of premature beats the incidence of rhythm and conduction defects in the entire population was 40 per cent. Table V represents the incidence of new arrhythmias secondary to myocardial infarction. The total is greater than 100 per cent as many patients had more than one type.

TABLE V

Arrhythmias	NO.	% of Total
Premature ventricular contractions	71	24
Premature atrial contractions	27	9
Premature nodal contractions	3	1
Atrial fibrillation	24	13
Supraventricular tachycardia	10	3
Ventricular tachycardia	5	2
Ventricular fibrillation	8	3
Intra-ventricular conduction defects	38	13
Atrio-ventricular conduction defects	31	10

A-V Block

Thirty-one patients (10.3 per cent) of the entire population developed atrio-ventricular conduction defects subsequent to their myocardial infarction. Of these, 21 were males and 10 females. The median age for this population was 70 years with a range between 30 and

85 years. The median age for males was 58 while the median age for females was 80. Eleven of 21 males (52.4 per cent) were under 60 years of age while only 1 of 10 females (10 per cent) was under 60. Two of 21 males (9.5 per cent) were over 70 while 8 of 10 females (80 per cent) were over 70.

The incidence of 3 types of atrio-ventricular conduction defects in relation to the entire population is outlined in Table VI.

TABLE VI

Type of Block	NO.	% of Total
1st degree block	10	3.3
1st degree and 2nd degree variable	8	2.6
3rd degree block	13	4.3

Patients in the first group showed no other manifestations of block other than a prolonged P-R interval (greater than .21 seconds). The second group included 3 patients who developed a Wenckebach phenomenon, but never went on to complete block. The third group included 8 patients with only complete atrio-ventricular dissociation and 5 patients who went through varying degrees of 1st degree and 2nd degree block before developing complete dissociation.

The mortality for this population was 9 of 31 (29 per cent). For males, the mortality was 7 0f 21 (33.3 per cent), for females, 2 of 10 (20 per cent). Within each of the 3 groups of block outlined in Table VI, the mortality was 3 of 10 (30 per cent) for 1st degree block, 1 of 8

(12.5 per cent) for variable block, and 5 of 13 (38.5 per cent) for complete block. There was 1 instance of sudden death without signs of congestive heart failure, shock or embolization.

A-V Block versus Tachyarrhythmias and "Control"

Thirty-four patients (11.3 per cent) developed tachyarrhythmias and 134 patients (44.7 per cent) developed no arrhythmia (the "control" group). The combined group of supraventricular tachyarrhythmias represents 24 patients with atrial flutter-fibrillation, 3 patients with paroxysinal atrial tachycardia (2 of whom also developed a nodal tachycardia) and 7 patients with an undefined supra-ventricular tachycardia.

Table VII represents demographic data for patients with A-V block, tachyarrhythmias and the "control" group.

TABLE VII

	A-V Block		Tachyarrhythmia		No Arrhythmia	
	NO.	%	NO.	%	NO.	%
Total Number	31	10.3	34	11.3	134	44.7
Male/Female	21/10		19/15		94/40	
Total Median Age	70		71		60	
M/F Median Age	58/80		63/74		59/61	

The mortality experience in these 3 groups, the incidence of sudden death and the length of hospitalization in days for survivors is outlined in Table VIII.

TABLE VIII

	A-V Block		Tachyarrhythmia		No Arrhythmia	
	NO.	%	NO.	%	NO.	%
Total Mortality	9	29	19	56	28	21
M/F Mortality	7/2	33/20	8/11	42/73	20/8	21/20
Sudden Death	1	3	3	9	7	5
Median Length of Hospitalization	23.5		21		21	

Cardiac history and status is shown in Table IX in relation to previous myocardial infarction and the prior incidence of congestive heart failure, hypertension, antecedent arrhythmias and the use of digitalis.

TABLE IX

Cardiac History	A-V Block		Tachyarrhythmia		No Arrhythmia	
	NO.	%	NO.	%	NO.	%
Previous M.I.						
1	7	23	7	21	32	24
More than 1	1	3	1	3	3	2
Questionable	1	3	0	-	6	5
Cardiac Status						
CHF	8	26	10	29	22	16
Hypertension	13	42	15	44	42	31
Antecedent Arrhy.	8	26	5	14	2	2
Medications						
Digitalis	9	29	12	35	24	18

Of the 199 patients under study in the above 3 populations, electrocardiographic evidence of myocardial infarction was present in 130 (65 per cent). Table X compares the distribution as to location of the various types of infarctions for those patients who had electrocardiographic evidence of infarction.

TABLE X

	A-V Block		Tachyarrhythmia		No Arrhythmia	
	NO.	%	NO.	%	NO.	%
Location of Infarct						
Anterior-lateral	2	10	4	27	29	31
Anterior-septal	5	25	4	27	20	20
Posterior-diaphragmatic	12	60	7	47	41	43
Posterior-lateral	1	5	-	-	3	3
Mixed anterior and posterior	-	-	-	-	2	2

A comparison of general morbidity among the 3 groups is shown in Table XI with respect to the development of congestive heart failure, shock, embolic phenomena and syncope subsequent to infarction.

TABLE XI

Morbidity	A-V Block		Tachyarrhythmia		No Arrhythmia	
	NO.	%	NO.	%	NO.	%
CHF	12	39	17	50	30	22
Shock	13	42	18	53	24	18
Embolic	3	10	5	14	3	2
Syncope	3	10	2	6	3	2

Table XII is a representation of the use of drugs in the respective groups which have been shown to affect cardiac rate and rhythm either directly or indirectly.

TABLE XII

Drugs	A-V Block		Tachyarrhythmia		No Arrhythmia	
	NO.	%	NO.	%	NO.	%
Digitalis	19	61	29	85	42	31
Diuretics	9	29	16	47	21	16
Pressors	14	45	18	53	21	16
Procaine Amide	3	10	6	18	0	-
Quinidine	2	7	6	18	0	-
Atropine	4	13	4	12	2	2
Steroids	4	13	1	3	0	-

IV

DISCUSSION

Total Population: Comparisons With Previous Studies

The differential incidence of myocardial infarction in males and females reported in the past has been confirmed by this study. Mintz and Katz[22] and Rosenbaum and Levine[26] have shown figures almost identical to the 67 per cent male and 33 per cent female incidence in this study. Comparable figures for age distribution are difficult to assess because most studies have used an average age which tends to be distorted by the incidence of infarction at the 2 age extremes. The median age of 63 years, however, for the entire population is slightly higher than the average age of 60.4 of Mintz and Katz.[22] Women who sustain a myocardial infarction tend to be older than the men. The higher median age of females of 70 as compared to that of 60.5 for the males is probably the greatest determinant of the fact that 38 per cent of females die during acute infarction and immediate hospital course as compared to 23 per cent of the males. Mintz and Katz[22] found a differential mortality of 28.9 per cent for women and 18.6 per cent for men. They attempted to explain the poorer prognosis in women on the basis of a higher incidence of diabetes mellitus, a greater frequency of thromboembolic phenomena and arrhythmias, in addition to their older age. Only the last parameter appears to be confirmed by this study. If diabetes mellitus were an important differential factor in mortality, one might also expect a concomitant increase in the percentage of women who sustain infarctions as compared to men.

The incidence of sudden death in the population, without congestive heart failure, shock or embolization, was 5 per cent. It has been assumed in the past that these deaths were probably due to sudden ventricular fibrillation or standstill. Woods and Barnes[31] have made this assumption using autopsy findings on 48 of 60 patients who died in their series of myocardial infarctions. Their figures show an incidence of death without apparent cause in 53 per cent of cases.

Among the important factors in the previous cardiac history, hypertension was present most often. Mintz and Katz[22] reported a variable incidence of between 28 per cent and 69 per cent. In this series, the incidence was 36 per cent using the criteria of a history of hypertension recorded in the chart or a blood pressure greater than 150 systolic or 100 diastolic during the present admission.

Fifty-one per cent of infarctions for which there was electrocardiographic evidence were of the anterior wall (both anterior-lateral and anterior-septal). Forty-eight per cent were of the posterior wall (diaphragmatic and posterior-lateral). Mintz and Katz[22] reported an incidence of 55 per cent for anterior infarction and 36 per cent for posterior infarction. Rosenbaum and Levine[26] also noted a higher incidence of anterior infarction, 50 per cent to 31 per cent.

General morbidity subsequent to myocardial infarction was assessed in relation to the occurrence of congestive heart failure, shock and embolic phenomena. Some degree of congestive heart failure was present in 34 per cent of the population. A range of 23 per cent to 70 per cent has been reported for all degrees from mild to severe.[26] Johnson and Miner,[16] however, found an incidence as low as 15.5 per cent. Shock was

present in 29 per cent of the patients, which compares with Rosenbaum and Levine's [26] finding of an incidence of 26 per cent. Johnson and Miner, [16] again however, found a lower incidence of only 14.5 per cent. Embolic phenomena were observed in 6 per cent of the total population. Mintz and Katz [22] reported a 9.9 per cent incidence. Woods and Barnes [31] found an incidence of 7.8 per cent for their total population with an incidence of 16.7 per cent in patients who died.

The use of standard preparations of digitalis, quinidine, procaine amide and other cardiac drugs has increased in recent years to control changes in rate and rhythm in the post-infarction period. The use of these drugs is not in complete favor. Master et al. [20] considered the use of digitalis, quinidine and epinephrine dangerous in the treatment of myocardial infarction because they felt that these drugs had too great a potential for initiating arrhythmias. They felt that the rarity of ventricular tachycardia in their series was due to the fact that digitalis was not used routinely. In this series digitalis was used in 48 per cent of patients, sympathomimetics in 26 per cent and quinidine and procaine amide in 12 per cent. Premature beats occurred in 34 per cent of cases whereas Master et al. [20] found an incidence of only approximately 25 per cent. The total incidence of arrhythmias of 40 per cent in this series falls in the middle range of the comprehensive study of Pick [25] who compiled an incidence for 3,174 cases of between 15 per cent to 73 per cent where disturbances of rate were also included.

A-V Block

The incidence of A-V block complicating myocardial infarction in this series was 10.3 per cent. As mentioned previously, a review of

1500 unselected cases by Master et al.[21] reported an incidence of between 0.7 per cent and 10 per cent for complete heart block (with an average less than 5 per cent) plus an additional incidence of from 2.5 per cent to 5.9 per cent for lower grades of block. Pick's[25] study recorded an incidence of between 1 per cent to 12 per cent for incomplete block plus an additional 1 per cent to 3 per cent for complete block. The high incidence in the present series can be explained by the fact that this series includes 1st degree block in the category of A-V conduction defects. These defects have in the past been considered benign without specific significance in terms of prognosis.[6,21] The impetus for including these patients in this group in this study has been the impression of Rosenbaum and Levine[26] that 1st degree block implies myocardial injury and that any degree of block may progress to another without warning. Indeed, it was found in this study that 13 patients (42 per cent) of the entire A-V conduction group who went on to develop higher grades of 2nd degree and 3rd degree block passed through a stage of 1st degree block. In addition, a comparative study of mortality for patients with only 1st degree block as compared to 2nd degree and 3rd degree block together, reveals an almost identical 30 per cent incidence in the two groups. Third degree block alone, however, showed the highest mortality of 38.5 per cent. A review of Table VI reveals that if the patients with only 1st degree block were eliminated, the incidence of partial and complete block would drop to 6.9 per cent which is more within the range determined in past studies.

The distribution of males and females in the A-V conduction group was similar to that of the entire population, 68 per cent males, and

32 per cent females. This finding is not consistent with that of Master et al.[21] who found a 2 to 4 times greater incidence of partial and complete heart block in females. The median age for the entire A-V conduction group was 70 years which was 7 years older than the median for the entire population. Master et al.[21] found an average age of 57 and 61 respectively for partial and complete block.

Although the median age for females was actually 10 years older in this group than in the total population, only 2 of 10 (20 per cent) females with A-V conduction defects died as compared to a mortality of 38 per cent for females in the entire population. The 2 females who died were 78 and 81 years old respectively, and both had developed only 1st degree block. One died 8 days after admission in shock having sustained an anterior-lateral infarction, her sixth infarction. The other, admitted in atrial fibrillation subsequently developed 1st degree block and died of shock and pulmonary embolization of an anterior-septal infarction. The mortality for males was 33 per cent as compared to 23 per cent for the total population. The 7 males who died ranged in age from 30 to 75 years. One had 1st degree block and died in shock; 1 had 1st degree and 2nd degree variable block and died in shock and congestive heart failure having gone through stages of ventricular tachycardia and ventricular fibrillation; 5 had 3rd degree block with 4 dying in congestive heart failure and 1 after a syncopal episode brought on by ventricular tachycardia and fibrillation.

A-V Block versus Tachyarrhythmias and "Control"

Table IX shows that 25.8 per cent of patients with A-V block have sustained 1 or more previous infarctions. There is no difference

between this incidence and that for the tachyarrhythmias and "control." Master et al.[20] have confirmed this finding as have Cohen et al.[6] There is, however, a greater incidence of congestive heart failure, hypertension and antecedent arrhythmias in the A-V group as compared to patients with no arrhythmias. The incidence of antecedent arrhythmias is also greater than in the tachyarrhythmia group. Antecedent arrhythmias in the A-V group included 2 instances of atrial fibrillation and varying forms of A-V conduction defects which were not, however, present on admission.

The incidence of prior congestive heart failure or hypertension was not appreciably greater than in the tachyarrhythmia group. Cohen et al.[6] recorded only the prior incidence of hypertension for patients with A-V block. Their incidence was 28 per cent as compared to 42 per cent in this series. They do not, however, define hypertension. The prior use of digitalis in the A-V group was 29 per cent. Cohen et al.[6] found only 13 per cent prior use. All their patients died during the acute illness. The incidence in this series was higher than in the "control" group, but not as high as in the tachyarrhythmia group.

Pick[25] found a range in mortality for incomplete A-V block from 20 per cent to 54 per cent and in complete block from 60 per cent to 100 per cent. This compares to the 20 per cent to 29 per cent overall mortality for recent infarction. In addition, the incidence of mortality for atrial fibrillation-flutter and other supraventricular ectopic tachycardias considered separately, has been respectively 18 per cent to 50 per cent, and 25 per cent to 60 per cent. In this series the overall mortality for the entire A-V group was 29 per cent. For 3rd

degree block alone, mortality was 38.5 per cent. Both these mortalities are higher than the mortality for patients with no arrhythmias which is only 21 per cent, but lower than patients with tachyarrhythmias whose mortality is 56 per cent.

The experience of Woods and Barnes,[31] cited previously, showed a greater than 50 per cent incidence of death without cause apparent at post-mortem examination. The assumption has been that these deaths have been caused by sudden ventricular fibrillation or standstill. Third degree A-V block is known to predispose to these conditions. Penton et al.[24] have reported an incidence of 12 per cent of sudden death in 49 patients who develop A-V block secondary to myocardial infarction. Table VIII shows that 1 of 31 patients (3 per cent) with A-V block died suddenly without congestive heart failure, shock or embolization. The incidence in the other 2 groups was 9 per cent for tachyarrhythmias and 5 per cent for the "control."

Of the 16 sudden deaths in the entire population, 7 (44 per cent) were in patients who developed no arrhythmia subsequent to myocardial infarction. Five were males ranging in age from 40 to 86 and 2 were females aged 67 and 83. Five patients died within the first 24 hours of hospitalization and 1 each on the third and fifth days respectively. There were no distinctive characteristics about these patients to distinguish them from the overall population with no arrhythmias. This experience confirms the impression that for any given patient who has sustained a myocardial infarction, prognosis during the early post-infarction period must be guarded.

The 1 patient with A-V block who died suddenly was a 58 year old

male who sustained a posterior-lateral infarction and developed complete heart block after progressing through stages of 1st degree and 2nd degree block. He experienced episodes of syncope and died on the fifth hospital day of ventricular fibrillation. The use of atropine, quinidine, procaine amide, prednisone, and the cardiovertor were all of no avail.

Two of 3 patients with tachyarrhythmias who died suddenly, died within 24 hours. Both had undefined supraventricular tachycardias and 1 had an associated intraventricular conduction defect. The third died on the 12th hospital day in atrial fibrillation without obvious change in her course. Of the remaining 5 sudden deaths in the entire population, 2 patients died of ventricular fibrillation, 1 of ventricular tachycardia. All failed to respond to external electrical stimulation. Two patients died without apparent reason, 1 with a nodal rhythm and 1 with an intraventricular conduction defect.

It has been the experience of all observers reviewing cases of complete heart block subsequent to myocardial infarction, that the predominant site of infarction is the posterior myocardium secondary to occlusion of the right coronary which supplies the A-V node in over 90 per cent of instances. Table X shows an incidence of 65 per cent posterior infarction (both posterior-diaphragmatic and posterior-lateral) for the A-V group which is appreciably higher than the 47 per cent incidence for the tachyarrhythmias and the 46 per cent incidence for the "control." Of the 13 documented posterior infarctions in the A-V group, 8 were in patients with 3rd degree block representing 8 of 9 patients (89 per cent) who had definite electrocardiographic evidence of infarction; 4 were in patients with variable block representing 4 of 5

patients (80 per cent) in this category; and 1 was in a patient with 1st degree block representing only 1 of 6 (17 per cent) in this group. It was the experience of Johnson and Miner[16] that the only one of 11 cases of A-V block in their series who did not sustain a posterior infarction was a patient with 1st degree block. It would thus appear from this study that although posterior infarction is more often the rule in variable and 3rd degree block, it is not so frequently the site of infarction in 1st degree block.

Secondary complicationsof congestive heart failure, shock, embolization and syncope have been shown to be increased in patients with varying types of arrhythmias complicating myocardial infarction.[1] Rosenbaum and Levine[26] showed a greater incidence of shock in patients with A-V block, although they did not find a greater incidence of congestive heart failure. Table XI shows that there is an increase in the occurrence of congestive heart failure, shock, embolization and syncope as compared to the "control." However, except for syncope, the incidence of congestive heart failure, shock and embolization is higher in the tachyarrhythmia group.

Some authors fear that the use of preparations such as digitalis, quinidine and sympathomimetics subsequent to infarction will increase the incidence of cardiac arrhythmias.[20] Others, feel that this fear is greatly overemphasized.[16] In most cases it was not possible in this study to relate the use of digitalis or other cardiac drugs to the onset of a specific arrhythmia. Five of 31 cases of 1st degree heart block, however, were in fact suspected as being secondary to digitalis intoxication. In addition, the use of digitalis in patients who developed

A-V block was twice that of patients who developed no arrhythmia. The high incidence of the use of digitalis in patients who develop A-V block suggests that digitalis may be an etiological factor. In no instance was digitalis used to treat A-V block. The use of sympathomimetic agents in heart block correlates well with the incidence of shock and is about 3 times as frequent in the A-V group as compared to the "control." Digitalis and pressors were, however, used more frequently in the tachyarrhythmia population as was quinidine and procaine amide. Neither of these substances was used in the "control" group. The use of digitalis and quinidine preparations is thus more frequent in the tachyarrhythmias but these drugs are still used in a high proportion of patients who develop A-V block.

V

SUMMARY AND CONCLUSIONS

The incidence of new A-V conduction defects subsequent to myocardial infarction was found to be 31 cases in a series of 300 myocardial infarctions (10.3 per cent). The median age for males was 58, and for females 80 years. First degree block (P-R interval greater than .21 seconds) occurred 10 times (3.3 per cent); 1st degree and 2nd degree variable block occurred 8 times (2.6 per cent) and 3rd degree block occurred 13 times (4.3 per cent). The mortality for the entire population of A-V conduction defects was 29 per cent with 3 of 10 deaths (30 per cent) in patients with 1st degree block; 1 of 8 deaths (12.5 per cent) in patients with 1st degree and 2nd degree variable block; and 5 of 13 deaths (38.5 per cent) in patients with complete block. There was 1 instance of sudden death without signs of congestive heart failure, shock or embolization.

Thirty-four patients (11.3 per cent) developed supraventricular tachyarrhythmias and 134 patients (44.7 per cent) developed no arrhythmia ("control"). The median age in the tachyarrhythmia group was 63 years for males and 74 years for females. For those with no arrhythmia the respective male and female median ages were 59 and 61 years. The total mortality for the tachyarrhythmia group was 56 per cent and 21 per cent for the "control" group. Sudden death occurred in 9 per cent of the tachyarrhythmias and in 5 per cent of the "control."

The following conclusions have been drawn from this study:

1) The incidence of A-V block complicating myocardial infarction

is higher than has previously been recorded. This is partially due to the fact that in this series 1st degree block is combined with higher degrees of block for comparative study. More important, however, is a more widespread use of digitalis preparations in the post-infarction period. Five of 31 cases of A-V block were in fact strongly suspected to be secondary to digitalis intoxication. The incidence of supraventricular tachyarrhythmias was approximately equal to that of A-V block. (See Tables VII and XII, pp. 14 and 17)

2) First degree block complicating myocardial infarction is not as benign an arrhythmia as has been considered in the past. Once an injury has been sustained by the myocardium, one degree of block may develop into another with a concomitant increase in mortality. (See p. 21)

3) There is a higher incidence of previous congestive heart failure, hypertension, antecedent arrhythmia and prior use of digitalis in the A-V group as compared to the "control." Antecedent arrhythmias are more common in the A-V group than in the tachyarrhythmias, whereas the prior use of digitalis is higher in the tachyarrhythmias. Posterior infarction is most common in the A-V group. (See Tables IX and X, pp. 15 and 16)

4) There is a greater number of secondary complications in patients with A-V block as compared to those with no arrhythmias. Except for syncope, however, secondary complications are even more common with the tachyarrhythmias. (See

Table XI, p. 16)

5) The mortality of patients with A-V block is not appreciably higher than for the "control." Mortality of patients with tachyarrhythmias, however, is much higher than for either of these 2 groups. Sudden death is unpredictable and is not limited to any one type of arrhythmia. (See Table VIII, p. 15)

6) Because the majority of patients with A-V block do not die suddenly of presumably electrical causes alone, an appreciable salvage rate with the use of the cathode pacemaker for A-V block is not likely. However, as the largest proportion of all patients who die suddenly expire within the first few hospital days, there is a potential for greater salvage of post-infarction patients with constant monitoring and surveillance of all patients, regardless of their clinical condition, during the first few initial hospital days. (See pp. 15 and 24)

VI

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